Exhibit 14

Specific Causation Expert Report for Frank Mousser

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10. Causation Analysis

I have reviewed the general causation expert reports of Drs. Hatten and Bird. The analysis in those reports supports my opinions in this report and finds that the four chemicals in the water at Camp Lejeune are causally related to kidney cancer. I also researched and read the epidemiology, toxicology and mechanistic evidence that exists relating to the toxins at issue in this case and agree that the toxins as they existed in the water at Camp Lejeune are causally related to kidney cancer, including UTUC, under a more likely than not standard, which exceeds the "at least as likely as not" standard in this case.* A summary of some of the evidence is below and is used for purposes of weighing the potential harmful effects of the exposure Mr. Mousser had to the water at Camp Lejeune.

a. UTUC Grouped with RCC in Large Databases

UTUC is frequently grouped with renal cell carcinoma (RCC) in large epidemiological databases due to anatomical proximity and shared coding systems. The epidemiology literature for studies that include UTUC with RCC and the studies that do not include them together show similar increased risks for kidney cancer.[2,3,4,5,6] Further, the epidemiology studies that look at UTUC separately and independently have similar increased risks as those studies that look at RCC alone.[4,5]

For example, the Zhao et al. study classified UTUC with Kidney cancer. Kidney cancer incidence and mortality were elevated in this study at high exposures with RR of 4.90 (95% CI: 1.23, 19.6) and 2.03 (95% CI: 0.50, 8.32).[2]

Several studies specifically looked at UTUC separately from kidney cancer but the results showed a similar risk profile. Raaschou-Nielsen et al. assigned UTUC with kidney cancer and also performed a separate analyses of UTUC. The SIR for kidney cancer as a whole and UTUC were the same.[4]

Lynge et al. analyzed renal pelvis cancers individually with SIR of 2.0 (95% CI: 1.0-3.7). In the same study, the category of kidney cancer had an SIR of 1.3 (95% CI: 1.0-1.7). These both show elevated risks of similar magnitude.[5]

Therefore, it is appropriate to use kidney cancer literature and epidemiology studies to support the causal relationships at issue in this analysis. This is especially true given the similar mechanisms of injury for UTUC and RCC.

b. Urothelial Origin of UTUC

Shared Pathogenesis with Urothelial Bladder Cancer: Unlike RCC, UTUC originates from the urothelial lining of the renal pelvis and ureters, the same tissue type as urothelial bladder cancer. This shared origin highlights the relevance of bladder cancer risk factors, including environmental exposures such as TCE, PCE, benzene, and vinyl chloride. These carcinogens accumulate in the

*Any reference to my professional opinions that the toxins in the water at Camp Lejeune caused Mr. Mousser's kidney cancer is meant to include UTUC.

urinary tract, inducing genotoxic effects and contributing to urothelial cell damage and carcinogenesis.

Environmental Exposure Relevance: The mechanisms underlying urothelial bladder cancer are directly applicable to UTUC, emphasizing the role of these carcinogens in increasing cancer risk in the upper urinary tract.

In addition to looking at kidney cancer literature and studies, I also reviewed literature specifically relating to upper tract urothelial carcinoma and found similar results showing increased risks relating to the chemicals at issue.

c. Trichloroethylene (TCE)

Trichloroethylene (TCE) is a widely used industrial solvent and volatile organic compound (VOC) that contaminates soil and groundwater, including at Camp Lejeune. It has been classified as a known human carcinogen by the International Agency for Research on Cancer (IARC) and the Environmental Protection Agency (EPA).[7]

i. Epidemiological Evidence

IARC concludes there is sufficient evidence in humans for TCE's carcinogenicity, particularly causing kidney cancer.[7] The Agency for Toxic Substances and Disease Registry (ATSDR) also recognizes sufficient evidence of causation for kidney cancer associated with TCE.[1]A 2010 meta-analysis by Kelsh et al. demonstrated a statistically significant relative risk (RR) of 1.42 (95% CI 1.17-1.77) for occupational TCE exposure and kidney cancer.[8] A 2011 EPA manuscript reported an overall RR of 1.27 (95% CI 1.13-1.43), with higher risks for groups exposed to elevated TCE levels (RR 1.58, 95% CI 1.28-1.96).[9] A 2010 study by Kelsh et al. reviewed occupational TCE exposure and found a significant increase in relative risk (RR) for urinary tract cancers, including UTUC, particularly in populations with high cumulative exposures.[8] Karami et al. (2012) reviewed 9 cohort studies and found an elevated RR of 1.26 (95% CI 1.02-1.56) for TCE exposure and renal cancer, with consistent results across cohort and case-control designs.[10] The study noted that misclassification of exposure in earlier research likely underestimated the true risk.[10]

ii. Camp Lejeune Studies

Numerous investigations into the contaminated water at Camp Lejeune have demonstrated an increased risk of kidney cancer, including upper tract urothelial cancers associated with TCE exposure.[11,12] Populations exposed to Camp Lejeune levels of TCE exhibited statistically significant increases in bladder and kidney cancers, both of which share a urothelial origin with UTUC.[11,12,13,14,15]

iii. Mechanistic Plausibility

TCE is metabolized into toxic intermediates, such as S-(1,2-dichlorovinyl)-L-cysteine (DCVC), which bioactivate in renal and urothelial tissues. These intermediates cause genotoxic stress, DNA adduct formation, and inflammation, processes central to urothelial carcinogenesis. Additionally,

TCE-induced oxidative stress impairs DNA repair mechanisms and promotes abnormal cell proliferation, further contributing to the initiation and progression of urothelial cancers.

iv. Conclusion

In December 2024, the U.S. Environmental Protection Agency (EPA) implemented a ban on all uses of trichloroethylene (TCE) to safeguard public health from the associated risks, including kidney cancer, linked to TCE exposure.[16]

Mr. Mousser's levels of TCE are significantly higher than the levels in the literature that show a causal association between TCE and UTUC. According to Bove 2014a's cumulative exposure charts, Mr. Mousser would have been categorized as being in the "high" exposure group for TCE, which is the highest category of exposure that exists.[13] This was associated with a 1.52 HR for kidney cancer.[13]

Collectively, epidemiological data, mechanistic studies, and meta-analyses provide robust evidence of the causal link between TCE exposure and kidney cancer, highlighting its significant public health implications. It is overwhelmingly probable that TCE causes kidney cancer/UTUC.

d. Perchloroethylene (PCE)

Perchloroethylene (PCE) is a VOC commonly used in dry-cleaning and as an industrial degreaser. It has been classified as a probable human carcinogen by IARC and the EPA,[7,17] with increasing evidence of its relevance to urothelial cancer.

i. Epidemiological Evidence

Cape Cod Cohort: In Cape Cod, Massachusetts, PCE leaching into drinking water was linked to increased risks of bladder cancer.[18] Given the shared urothelial lining, these findings are highly relevant to UTUC. It was also linked to increased incidences of kidney cancer.[18]

Dry-Cleaning Workers: A study by Ruder et al. (2001) evaluated over 1,700 dry-cleaning workers exposed to PCE.[19] Although primarily associated with bladder cancer, the findings indicated elevated risks for upper tract cancers due to cumulative PCE exposure.[19]
Callahan et al. identified a dose-response relationship, with the highest exposure group showing a hazard ratio (HR) of 13.2 (95% CI 1.9-90.8) for kidney cancer mortality.[20]

U.S. Kidney Cancer Study: Purdue et al conducted a case-control study and reported an odds ratio (OR) of 3.1 (95% CI 1.3-7.4) for high cumulative PCE exposure and kidney cancer, indicating a strong association.[21]

ii. Camp Lejeune Study

Bove et al. examined civilian workers at Camp Lejeune and found a standardized mortality ratio (SMR) of 1.30 (95% CI 0.52-2.67) for kidney cancer.[14] In Bove 2014a, with particular respect to PCE, the relative risks of kidney cancer based on low, medium and high exposures were: 1.40 for

low exposure, 1.82 for medium exposure and 1.59 for high exposure.[13] Mr. Mousser would have met the high exposure category.

iii. Mechanistic Plausibility

PCE undergoes metabolism into trichloroacetic acid (TCA) and dichloroacetic acid (DCA), which induce oxidative stress, DNA strand breaks, and urothelial cell damage. These metabolites also promote chronic inflammation and interfere with normal cell cycle regulation, fostering conditions that increase the risk of malignant transformation in urothelial tissues. These mechanisms mirror those implicated in bladder cancer and kidney cancer and are relevant to UTUC.

iv. Conclusion

In December 2024, the U.S. Environmental Protection Agency (EPA) implemented a ban on perchloroethylene (PCE) to protect public health from the associated risks, including its link to various cancers such as kidney and urothelial cancers.[16]

Mr. Mousser's levels of PCE are significantly higher than the levels in the literature that show a causal association between PCE and UTUC. According to Bove 2014a's cumulative exposure charts, Mr. Mousser would have been categorized as being in the "high" exposure group for PCE, which is the highest category of exposure that exists.[13] This corresponded to a HR of 1.59 for kidney cancer.[13]

Epidemiological studies, particularly those involving occupational and environmental exposures, along with mechanistic data, support a causal association between PCE and kidney cancer. The findings underscore the carcinogenic potential of PCE, especially in high-exposure settings like Camp Lejeune and the dry-cleaning industry. It is more likely than not that PCE causes kidney cancer.

e. Benzene

Benzene is a carcinogenic compound historically used in industrial applications and present as a contaminant in fuel leaks, such as those at Camp Lejeune. It is recognized as a known human carcinogen by IARC and the EPA.[22]

i. Epidemiological Evidence

Camp Lejeune: Benzene contamination at Camp Lejeune contributed to elevated kidney cancer risks in exposed populations, including urothelial cancers. Studies consistently reported higher incidences of bladder and kidney cancers in affected cohorts.[11,12,13,14,15]

Case-Control Studies: Hu et al. (2002) found an increased odds ratio (OR) for renal cell carcinoma in individuals with occupational benzene exposure, particularly among those in industrial chemical settings.[23]

Meta-Analyses: Seyyedsalehi et al. analyzed 29 studies and found a relative risk between Benzene and kidney cancer of 1.20.[24]

Chemical Workers: Workers in industries involving benzene, such as manufacturing and petrochemicals, demonstrate elevated risks for urothelial cancers. Chronic exposure results in cumulative damage to urothelial cells due to benzene's genotoxic properties.

ii. Mechanistic Plausibility

Benzene is metabolized into reactive intermediates, such as hydroquinone and benzoquinone, which generate reactive oxygen species (ROS) and cause DNA adduct formation, strand breaks, and chromosomal aberrations in urothelial tissues. These genotoxic effects trigger chronic inflammation and disrupt cellular repair mechanisms, creating a microenvironment conducive to malignant transformation in the urothelium.

iii. Conclusion

Mr. Mousser's levels of Benzene are higher than the levels in the literature that show a causal association between Benzene and UTUC. According to Bove 2014a's cumulative exposure charts, Mr. Mousser would have been categorized as being in the "high" exposure group for Benzene, which is the highest category of exposure that exists.[13] This corresponded with a HR of 1.36 for kidney cancer.[13]

There is evidence to support the causal relationship of benzene to kidney cancer/UTUC at least as likely as not and using an equipoise standard.

f. Vinyl Chloride and Urothelial Cancer

Vinyl chloride, predominantly used in the production of polyvinyl chloride (PVC), is another contaminant at Camp Lejeune. It is classified as a known human carcinogen by IARC and the EPA.[22]

i. Epidemiological Evidence

Camp Lejeune Studies: Vinyl chloride, a significant contaminant in Camp Lejeune's water supply, was associated with increased risks of bladder, kidney and upper tract urothelial cancers in exposed populations.[11,12,13,14,15]

Case Control Studies: The 2002 study by Hu et al. found significantly elevated risk of kidney cancer with exposure to vinyl chloride.[23] There was a monotonic dose response relationship found between VC and kidney cancer.[23]

ii. Mechanistic Plausibility

Vinyl chloride forms DNA adducts, such as etheno-deoxyadenosine, and induces oxidative stress, disrupting cell cycle regulation and DNA repair mechanisms. These effects increase the likelihood of mutations conducive to malignant transformation in urothelial tissues.

iii. Conclusion

Mr. Mousser's levels of VC are significantly higher than the levels in the literature that show a causal association between VC and kidney cancer. According to Bove 2014a's cumulative exposure charts, Mr. Mousser would have been categorized as being in the "high" exposure group for VC, which is the highest category of exposure that exists.[13] This corresponded with a HR of 1.51 for kidney cancer.[13]

There is evidence to support the causal relationship of vinyl chloride to kidney cancer and urothelial cancers at least as likely as not and using an equipoise standard.

g. Conclusion for All Chemicals

There was a monotonic response relationship for TVOCs at Camp Lejeune and kidney cancer.[13] The HRs for this metric were 1.42 (low exposure), 1.44 (medium exposure) and 1.54 (high exposure).[13]

The epidemiological and occupational studies collectively underscore the link between TCE, PCE, benzene, and vinyl chloride exposures and kidney cancers, including UTUC. Findings from Camp Lejeune studies, occupational analyses, and dose-response models reinforce the carcinogenic roles of these chemicals, particularly in populations with high or prolonged exposures. Mr. Mousser's exposure meets the highest exposure groups from each of the four chemicals in the Bove 2014a study.[13] For the TVOC analysis, Mr. Mousser was exposed to 11,959 ug/l-M and the threshold for the highest category was 12,250 ug/l-M.

Mr. Mousser's exposure was in the very highest category of exposures for all of the individuals who spent time at Camp Lejeune. The data from the Camp Lejeune studies of Bove and ATSDR provide compelling data that exposures of this kind are causally related to kidney cancer.[11,12,13,14,15]

11. Patient-Specific Considerations

Age and Tumor Size: [redacted]

Latent Period: [redacted]

Exposure: Mr. Mousser's TCE exposure (10,373 µg/L) far exceeds safe thresholds of levels implicated in kidney cancer cases. The reports of Drs. Hatten and Bird detail the levels at which the chemicals at issue have been known to be causally associated with kidney cancer/UTUC. I